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QR in V1 – an ECG sign associated with right ventricular strain and adverse clinical outcome in pulmonary embolism

Nils Kucher*, Nazan Walpoth, Kerstin Wustmann, Markus Noveanu, Marc Gertsch

Cardiology, Swiss Cardiovascular Center Bern, University Hospital, 3010 Bern, Switzerland

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Aims To test the hypothesis that Qr in V_1 is a predictor of pulmonary embolism, right ventricular strain, and adverse clinical outcome.
Methods and Results ECG's from 151 patients with suspected pulmonary embolism were blindly interpreted by two observers. Echocardiography, troponin I, and pro-brain natriuretic peptide levels were obtained in 75 patients with pulmonary embolism. Qr in V_1 (14 vs 0 in controls; $p<0.0001$) and ST elevation in V_1 ≥1 mV (15 vs 1 in controls; $p=0.0002$) were more frequently present in patients with pulmonary embolism. Sensitivity and specificity of Qr in V_1 and T wave inversion in V_2 for predicting right ventricular dysfunction were 31/97% and 45/94%, respectively. Three of five patients who died in-hospital and 11 of 20 patients with a complicated course, presented with Qr in V_1. After adjustment for right ventricular strain including ECG, echocardiography, pro-brain natriuretic peptide and troponin I levels, Qr in V_1 (OR 8.7, 95%CI 1.4–56.7; $p=0.02$) remained an independent predictor of adverse outcome.
Conclusions Among the ECG signs seen in patients with acute pulmonary embolism, Qr in V_1 is closely related to the presence of right ventricular dysfunction, and is an independent predictor of adverse clinical outcome.

Introduction

Electrocardiography (ECG) in patients with pulmonary embolism may show several abnormalities related to right ventricular strain. Many ECG signs are more frequent in patients with pulmonary embolism compared to those in whom pulmonary embolism is suspected but excluded, but none of the different ECG signs have been shown to be sufficiently specific to establish the diagnosis. In combination with a high clinical pretest probability or echocardiographic signs of right ventricular dysfunction, accuracy of ECG to diagnose pulmonary embolism may be improved.[1,2] On the other hand, ECG may be entirely normal in up to 20% of patients with pulmonary embolism resulting in a low sensitivity for the exclusion of the diagnosis.[3] However, ECG is obtained in almost all patients who present with dyspnoea, chest pain or syncope. Thus, its diagnostic value for patients with suspected pulmonary embolism is important even in the era of modern diagnostic strategies including spiral computed tomography (CT) and echocardiography.

Many years ago, Weber and Phillips observed a pseudoinfarction pattern with Q waves in lead V_1 in

* Corresponding author: Nils Kucher, MD, Cardiology, Harvard Medical School, Brigham and Women's Hospital, VTE Research Group, 75 Francis Street, Boston, MA 02115, USA. Tel.: +1-617-732-6986; fax: +1-617-738-7652
E-mail address: nkucher@freesurf.ch (N. Kucher).

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10 of 60 patients with pulmonary embolism.[3] This pattern was also found in 11 of 90 patients with pulmonary embolism in a later study.[4] It is assumed that this sign is caused by massive right-heart dilatation rotating the QRS vector away from the V_1-position resulting in a Q wave. We aimed to test the hypothesis that Qr in V_1 is a the predictor of pulmonary embolism, right ventricular strain, and adverse clinical outcome.

Methods

Patients

A total of 151 consecutive patients from a diagnostic and treatment study with suspected symptomatic pulmonary embolism from the emergency department were enrolled in the present analysis.[5] The study protocol was approved by the local ethics committee, and written informed consent was obtained from all patients.

Diagnostic strategy

The diagnostic strategy included D-dimer testing, assessment of clinical pretest probability according to the Wells criteria,[6] and spiral computed tomography (CT). In 30 patients with a D-dimer level below the cut-off value (500 ng/ml) and a low pretest probability, pulmonary embolism was excluded. In the remaining 121 patients with elevated D-dimer, including two patients with a negative D-dimer but a moderate or high pretest probability, spiral CT was performed. Pulmonary embolism was confirmed in 75 patients by spiral CT. In 34 patients with a negative spiral CT and a low clinical pretest probability, pulmonary embolism was excluded. In 12 patients with a negative spiral CT but a moderate or high pretest probability, further diagnostic tests were obtained: ventilation perfusion scan in six, compression sonography of the leg veines in four, and pulmonary angiography in two patients. According to the strategy, 75 patients were positive and 76 negative for pulmonary embolism.

Discharge diagnoses of the 76 patients without pulmonary embolism were musculoskeletal pain (n=22), pneumonia (n=17), chronic obstructive lung disease (n=15), congestive heart failure (n=14), acute coronary syndrome (n=2), pericardial effusion (n=2), aortic dissection (n=2), and sepsis (n=2). Three-month follow-up was performed in the 76 patients with initially negative test results. There was no venous thromboembolism among these patients, including 70 patients who received no anticoagulation during follow-up.

Laboratory tests

D-dimer was measured in all patients on admission using VIDAS D-dimer (bio-Mérieux, France), a quantitative enzyme-linked immunoassay automated on a VIDAS immunoanalyzer.[7] Troponin I and N-terminal pro brain natriuretic peptid (proBNP) levels were taken in all 75 patients with pulmonary embolism on admission. Troponin I was measured using a microparticle enzyme immunoassay (Abbott, USA), and proBNP with the Elecsys 2010 immunoassay analyzer (Roche, Germany). A Troponin I level ≥0.6 ng/ml was considered to be elevated.[8]

Spiral computed tomography (CT)

Pulmonary embolism was diagnosed by spiral CT when there was at least one intravascular filling defect in a pulmonary artery using a standard protocol.[9] A single-slice CT scanner Somatom Plus 4 UFC (Siemens, Erlangen, Germany) was used in 80 patients, and a multislice scanner Asteion MS (Toshiba, Tokyo, Japan) in 41 patients. The scan protocol with the single slice scanner included early arterial phase scanning 15–20 s after power-injection of a bolus of 120 ml of a contrast agent with 300 mg/ml iodine content using a 3 mm collimation, pitch 1.5, and a 2 mm reconstruction interval with coverage of the lung vasculature, followed by a second portal venous phase scan with 8 mm collimation and 8 mm reconstruction interval, pitch 1.5, covering the entire chest. The standard protocol of the multislice scanner included arterial phase scanning 20 s after power-injection of a 120 ml bolus of the contrast agent with 4|31 mm collimation, pitch 5.5/4 and 1 mm reconstruction interval with coverage of the entire chest. The interobserver agreement between two radiologists for spiral CT results was 97% (kappa 0.95, p<0.0001).

Transthoracic echocardiography

Echocardiography was performed in all patients with pulmonary embolism using an Acuson Sequoia™ C256 system (Mountain View, California, USA) with a 3.5 MHz probe and 3-lead electrocardiographic monitoring within 4 h after admission. Echocardiographic off-line analysis by soft-copy reading was performed by a cardiologist who was unaware of clinical data. Systolic right ventricular dysfunction was diagnosed in the

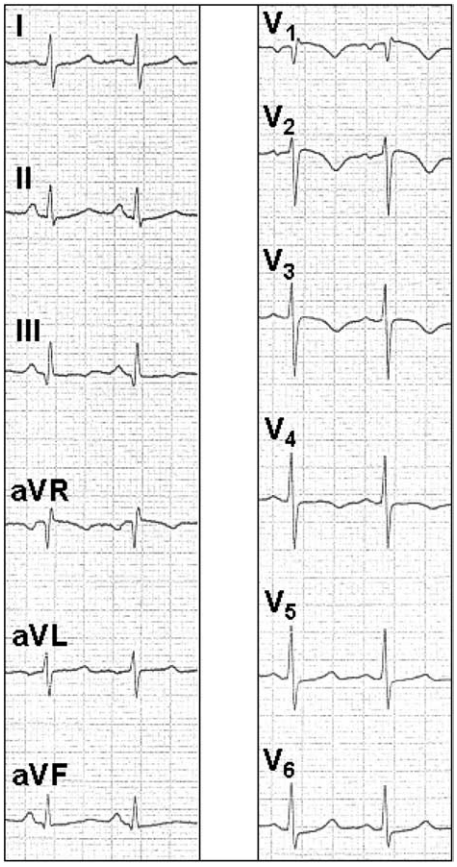

Fig. 1 Twelve-lead ECG from a patient with acute pulmonary embolism showing several signs of right ventricular strain: Qr in V_1, S_1Q_3, T wave inversion in V_2, QRS axis >50°, clockwise rotation of the QRS vectors in the precordial leads, heart rate of 100 beats per minute.

presence of moderately to severely depressed right ventricular free wall kinesis.[10,11]

Electrocardiography (ECG)

Twelve-lead ECG was obtained in all 151 patients with suspected pulmonary embolism at admission. ECG's were independently interpreted by two observers blinded to patient data. Following ECG signs were investigated: Heart rate >100 beats per minute, rightward shift of QRS axis >50°, Qr in V_1, S_1 subtypes ($S_1Q_3/S_1rSr'_3/S_1S_2S_3$), incomplete and complete right bundle branch block (RBBB), clockwise rotation of the QRS vector in the precordial leads (CLOCKROT) defined as R=S in V_4, V_5 or V_6, T wave inversion in V_2 or V_3 ($T_{NEG}V_2$), ST elevation in $V_1 \geq 0.1$mV ($ST_{POS}V_1$), and atrial fibrillation or flutter.[3,4,12,13] The criteria for the diagnosis of Qr in V_1 were the presence of a prominent Q wave of ≥0.2 mV and a ventricular depolarisation <120 ms (Fig. 1).

Table 1 Frequency of ECG signs in patients with and without pulmonary embolism

ECG sign	PE+ (*n*=75)	PE− (*n*=76)	SE (%)	SP (%)	NPV (%)	PPV (%)
Heart rate >100 bpm‡	28	13	37	83	44	68
Qr in V_1*	14	0	19	100	55	100
$S_1Q_3/S_1rSr'_3/S_1S_2S_3$†	50	28	67	50	40	64
Incomplete RBBB‡	21	8	28	89	41	72
CLOCKROT	31	21	41	72	44	60
$T_{NEG}V_2$‡	21	9	26	88	41	70
$ST_{POS}V_1$†	15	1	29	99	44	94
RBBB	2	4	3	95	51	33
QRS axis >50°	20	15	27	80	52	57
Atrial fibrillation	3	4	4	95	50	43
Atrial flutter	1	3	1	96	50	25

NPV=negative predictive value; PPV=positive predictive value; SE=sensitivity; SP=specificity. CLOCKROT=clockwise rotation of the QRS vector in the precordial leads; RBBB=right bundle branch block; $ST_{POS}V_1$=ST elevation ≥0.1 mv in V_1; $T_{NEG}V_2$=T wave inversion in V_2.

*$p<0.0001$
†$p<0.001$
‡$p<0.05$.

Statistical analysis

Kappa measurement was performed to examine interobserver agreement for ECG signs and spiral CT findings, respectively. Stepwise logistic regression was performed with univariately significant ECG signs (Table 1) to find the best model to predict or exclude pulmonary embolism. ProBNP levels in the presence or absence of ECG signs were compared with the Mann–Whitney u-test. Multivariate logistic regression analysis was performed to investigate the accuracy of ECG signs for predicting 'escalation of therapy' defined as the need for cardiopulmonary resuscitation, mechanical ventilation, pressors, thrombolysis, catheter fragmentation, or surgical embolectomy according to the MAPPET-3 criteria.[14] Following univariately significant variables were included in the multivariate analysis: Qr in V_1, $T_{NEG}V_2$, Troponin I ≥0.06 ng/ml, proBNP ≥500 pg/ml, and moderate to severe right ventricular systolic dysfunction. Differences in mortality and 'escalation of therapy' according to the presence or absence of ECG signs were analyzed using Fisher's exact two-sided test.

Results

ECG and pulmonary embolism diagnosis

Interobserver agreement was accurate for most ECG signs (Table 2). Agreement for the presence or

Table 2 Interobserver agreement for ECG findings in patients with suspected pulmonary embolism

ECG sign	Agreement (%)	Kappa*
Qr in V_1	98.7	0.92
$S_1Q_3/S_1rSr'_3/S_1S_2S_3$	89.4	0.79
Incomplete RBBB	90.1	0.68
RBBB	98.7	0.83
CLOCKROT	85.4	0.66
$T_{NEG}V_2$	92.1	0.71
$ST_{POS}V_1$	99.3	0.97
QRS axis >50°	93.2	0.73
Atrial fibrillation	100.0	1.00
Atrial flutter	100.0	1.00

*p<0.0001 for all kappa measurements. CLOCKROT= clockwise rotation of the QRS vector in the precordial leads; RBBB=right bundle branch block; $ST_{POS}V_1$=ST elevation ≥0.1 mv in V_1; $T_{NEG}V_2$=T wave inversion in V_2.

absence of S_1 subtypes and clockwise rotation of the QRS vector in the precoardial leads was moderately accurate.

None of the patients with excluded pulmonary embolism had a Qr in V_1 resulting in a specificity and sensitivity of 100% and 19%, and a positive and negative predictive value of 100% and 55%, respectively. Sinus tachycardia, S_1 subtypes, incomplete RBBB, $T_{NEG}V_2$, and $ST_{POS}V_1$ were more frequently present in patients with pulmonary embolism (Table 1). None of the ECG signs was sufficiently sensitive to exclude pulmonary embolism. The accuracy to predict or exclude pulmonary embolism was improved by the combination of Qr in V_1, $ST_{POS}V_1$, S_1 subtypes, and incomplete RBBB resulting in a specificity and sensitivity of 91% and 47%, respectively.

ECG signs in relation to right ventricular strain and clinical outcome in patients with pulmonary embolism

Moderate to severe right ventricular systolic dysfunction by echocardiography was present in 42 patients with pulmonary embolism. Elevated Troponin I levels were found in 25 patients with pulmonary embolism. Nine of 14 patients with Qr in V_1 and 10 of 15 patients with $ST_{POS}V_1$ had a troponin I level ≥0.6 ng/ml (Table 3). Significant differences in proBNP levels were found for Qr in V_1, iRBBB, CLOCKROT, and $T_{NEG}V_2$ (Fig. 2). Qr in V_1 and $T_{NEG}V_2$ were most specific for the prediction of right ventricular dysfunction, and S1 subtypes most sensitive for the exclusion of right ventricular dysfunction compared with other ECG signs (Table 4).

In patients with a shock index ≥1 (heart rate divided by systolic blood pressure) and right ventricular dysfunction, thrombolysis was performed in six and surgical embolectomy in four patients. Thrombolysis was also administered in six patients with a shock index <1 and right ventricular dysfunction. Heparin alone was given in the remaining 59 patients of whom 26 had right ventricular dysfunction but contraindications for thrombolysis according to the guidelines of the European Society of Cardiology.[15]

Five of the 75 patients with pulmonary embolism died in-hospital from right ventricular failure. Twenty patients had 'escalation of therapy' including the need for at least one of the following: cardiopulmonary resuscitation (n=3), mechanical ventilation (n=4), pressors (n=7), thrombolysis (n=12), embolectomy (n=4). In patients with 'escalation of therapy', systolic blood pressure was lower (89±27 vs 122±19 mmHg; p=0.011), and heart rate higher (114±20 vs 85±17 beats per minute; p=0.023) compared to patients without the need for 'escalation of therapy'.

Qr in V_1 was present in three (60%) of the five patients who died from right ventricular failure, and it was present in only 11 of 67 (16%) surviving patients (Table 5). Compared with other ECG signs, sinus tachycardia, Qr in V_1 and $T_{NEG}V_2$ also predicted 'escalation of therapy'. Sensitivity, specificity, negative and positive predictive value of Qr in V1 for predicting 'escalation of therapy' were 55%, 95%, 85% and 79%, respectively. In a multivariate regression analysis including the ECG signs Qr in V_1 and $T_{NEG}V_2$, results of echocardiography and biomarker tests, Qr in V1 (OR 8.7, 95%CI 1.4–56.7; p=0.02), proBNP levels ≥500 pg/ml (OR 12.5, 1.2–135.2; p=0.04), and Troponin I levels ≥0.06 ng/ml (OR 7.7, 1.6–36.5; p=0.01) remained independent predictors of adverse clinical outcome. Sensitivity, specificity, negative and positive predictive value of the combination of these prognostic markers for the prediction of 'escalation of therapy' were 75%, 91%, 75% and 91%, respectively.

DISCUSSION

ECG and pulmonary embolism diagnosis

The present study of 151 patients with suspected PE confirms previous reports that ECG alone is not sufficient to exclude pulmonary embolism.[2–4,16] Sensitivity and specificity of ECG to predict pulmonary embolism even in combination of different ECG signs (i.e. Qr in V_1, $ST_{POS}V_1$, S_1 subtypes, and incomplete RBBB) was 47% and 91%, respectively. The prevalence of Q waves in V_1 among patients with pulmonary embolism in the present study

Table 3 ECG signs according to the presence or absence of a Troponin I leak in 75 patients with pulmonary embolism

ECG sign positive*	Troponin I≥0.6 ng/ml(*n*=25)	Troponin I<0.6 ng/ml(*n*=50)	*p*
Heart rate >100 bpm	13(52)	15(39)	ns
Qr in V_1	9(36)	5(19)	0.008
$S_1Q_3/S_1rSr'_3/S_1S_2S_3$	19(76)	31(62)	ns
Incomplete RBBB	10(40)	11(22)	ns
CLOCKROT	15(65)	16(32)	0.01
$T_{NEG}V_2$	11(48)	10(20)	0.03
$ST_{POS}V_1$	10(40)	5(10)	0.002
QRS axis >50°	10(40)	10(20)	0.05

*For abbreviations see footnote in Table 1

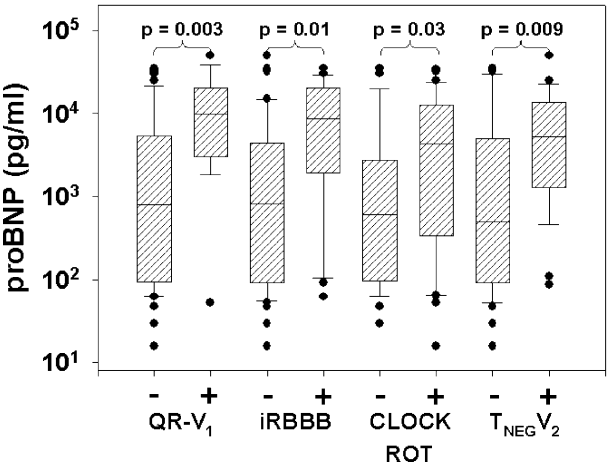

Fig. 2 Box plots showing logarithmic pro-brain natriuretic peptide levels in the presence or absence of ECG signs in 75 patients with pulmonary embolism. Boxes with error bars represent median values, 10th, 25th, 75th, and 90th percentiles.

(19%) was similar in the study of Weber et al. (17%) and Stein et al. (11%).[3,4] Due to a lack of a control group in the above mentioned studies, no statement about the diagnostic accuracy of this ECG sign was possible. In the present study, none of the 76 patients with excluded PE presented with a Qr in V_1 resulting in a specificity and positive predictive value of 100%, respectively.

ECG signs in relation to right ventricular strain and clinical outcome in patients with pulmonary embolism

The relation of ECG signs indicating right ventricular strain with haemodynamic data is well documented.[4,17–19] In 90 patients with pulmonary embolism, those with pseudoinfarction pattern (29±3 mmHg) and right axis deviation (31±7 mmHg) had highest mean pulmonary artery pressure values.[4] In a study of 80 patients with pulmonary embolism, $T_{NEG}V_2$ was associated with severely increased mean pulmonary artery pressure (37±8 mmHg) compared with the other ECG signs.[17] In the present study, QR in V_1 and $T_{NEG}V_2$ were most closely related to the presence of moderate to severe right ventricular dysfunction.

Pulmonary artery pressure is probably not the best outcome predictor because it depends on dynamics of embolic events, right ventricular systolic function and volume status. Thus, patients with chronic recurrent pulmonary embolism will probably tolerate higher pulmonary artery pressure values than those with a single massive embolic event. Novel and probably more accurate predictors of right ventricular dysfunction include markers of minor myocardial injury (cardiac troponins) and increased myocardial shear stress (b-type and atrial natriuretic peptides).[20–23] In the present study, $ST_{POS}V_1$ and Qr in V_1 were the only ECG signs associated with the presence of a troponin I leak. High proBNP levels were found in patients with Qr in V_1, $T_{NEG}V_2$, incomplete RBBB, and clockwise rotation of the QRS vector in the precordial leads.

Qr in V_1 was associated with increased early mortality. Three of five patients who died in-hospital from right ventricular failure initially presented with this sign. In addition, Qr in V_1 and $T_{NEG}V_2$ were highly associated with an increased risk of 'escalation of therapy' including the need for cardiopulmonary resuscitation, mechanical ventilation, pressors, thrombolysis, or embolectomy. After adjustment for the emerging markers of right ventricular strain including ECG signs, echocardiographic findings, troponin I and proBNP levels, Qr in V1 remained an independent predictor of 'escalation of therapy'.

Despite interpretation of negative spiral CT scans in context with clinical pretest probability, and the absence of venous thromboembolism among 46 patients with initially negative test results during follow-up, pulmonary embolism could have been present in a few cases. This might have

Table 4 Accuracy of ECG signs to predict moderate to severe right ventricular systolic dysfunction in 75 patients with PE

ECG Sign	RVD+(*n*=42)	RVD−(*n*=33)	SE(%)	SP(%)	NPV(%)	PPV(%)
Heart rate >100 bpm‡	20	8	48	76	53	71
Qr in V_1†	13	1	31	97	48	93
$S_1Q_3/S_1rSr'_3/S_1S_2S_3$*	35	15	83	55	72	70
Incomplete RBBB‡	16	5	38	85	48	76
$T_{NEG}V_2$*	19	2	45	94	57	90
$ST_{POS}V_1$‡	12	3	29	91	50	80

CLOCKROT, RBBB, QRS axis >50°, Atrial fibrillation and flutter were not different between patients with and without moderate to severe right ventricular dysfunction. NPV=negative predictive value; PPV=positive predictive value; RVD=moderate to severe right ventricular systolic dysfunction; SE=sensitivity; SP=specificity. For other abbreviations see footnote in Table 1

†$p<0.01$
*$p<0.001$
‡$p<0.05$.

Table 5 ECG signs and clinical outcome in 75 patients with pulmonary embolism

ECG sign positive	In-hospital death		*p*	Escalation of Therapy*		*p*
	Yes (*n*=5)	No (*n*=67)		Yes (*n*=20)	No (*n*=52)	
Heart rate >100 bpm	3	25	0.37	14	14	0.001
Qr in V_1	3	11	0.047	11	3	<0.0001
$S_1Q_3/S_1rSr'_3/S_1S_2S_3$	4	46	0.59	16	34	0.27
Incomplete RBBB	2	19	0.62	8	13	0.25
CLOCKROT	3	28	0.65	12	19	0.11
$T_{NEG}V_2$	3	18	0.14	12	9	0.0009
$ST_{POS}V_1$	3	12	0.06	8	7	0.02
QRS axis >50°	2	18	0.61	7	13	0.78

*Defined as the need for thrombolysis, embolectomy, mechanical ventilation, pressors or cardiopulmonary resuscitation. For abbreviations see footnote in Table 1

caused overestimation of sensitivity of the ECG signs, thus making it even lower than reported in the present study.

Conclusion

Qr in V_1 is highly specific for pulmonary embolism although its prevalence among consecutive patients with suspected pulmonary embolism is low. Compared with other ECG signs, Qr in V_1 is the strongest predictor of right ventricular dysfunction, and it is highly associated with troponin leakage and myocardial shear stress. Qr in V_1 and the presence of negative T waves in V_2 or V_3 also predict a complicated hospital course and therefore, are useful for risk stratification in pulmonary embolism. In patients with suspected acute pulmonary embolism, presenting with one of these ECG signs, more intensive hospital resources should be allocated to allow for a rapid diagnostic approach including echocardiography to confirm right ventricular dysfunction.

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